EXHIBIT D

30435.54USU1 SBA/RDG

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

Applicants	:	Robert Reiter and Owen Witte	
Serial No.	:	09/038,261	Examiner: Dr. Larry Helms
Filed	:	March 10, 1998	Group Art Unit: 1642
For	:	PSCA: PROSTATE STEM CELL ANTIGEN AND USES THEREOF	

35 N. Arroyo Parkway
Pasadena, California 91103

Assistant Commissioner for Patents
Washington, D.C. 20231

SIR:

**DECLARATION BY
ROBERT REITER AND OWEN WITTE
UNDER 37 C.F.R. §1.132**

We, Robert Reiter and Owen Witte, hereby declare that:

1. The assignee of record of the subject application is the University of California at Los Angeles (UCLA), in Los Angeles, California.

2. I, Robert Reiter, began my period of employment with UCLA in 1995 and presently hold the title of Assistant Professor in the Department of Urology. Additionally, I am a Co-Director of the Prostate Cancer Program at the Jonsson Comprehensive Cancer Center. I was employed by UCLA at the time of the invention.

3. I, Owen Witte, began my period of employment at UCLA in 1980 and presently hold the title of Professor in the Department of Microbiology, Immunology, and Molecular Genetics at UCLA. In addition, in 1986, I began a joint position as Investigator of the Howard Hughes Medical Institute (HHMI) at UCLA. I was employed by the HHMI at UCLA at the time of the invention.

4. We declare that we are the inventors of the claimed inventions: (1) a method for inhibiting the growth of prostate tumor cells expressing Prostate Stem Cell Antigen (PSCA) comprising administering to a patient a monoclonal antibody designated ATCC No. HB-12612, ATCC No. HB-12616, ATCC No. HB12618, or ATCC No. HB-12617 which binds specifically to the extracellular domain of PSCA in an amount effective to inhibit growth of the prostate tumor cells (e.g., claims 44-47); and (2) a method for selectively killing a cell expressing PSCA comprising reacting a monoclonal antibody designated ATCC No. HB-12612, ATCC No. HB-12616, ATCC No. HB12618, ATCC No. HB-12617 conjugated to a therapeutic agent with the cell so that the therapeutic agent conjugated to the antibody can kill the cell (e. g., claims 48).

5. We provide post-filing confirmatory support for the methods of inhibiting the growth of prostate tumor cells expressing PSCA, as claimed in claim 44 (see Exhibits 1-13).

6. Exhibit 1 depicts experimental results showing that administering an anti-PSCA monoclonal antibody to animals injected with prostate tumor cells expressing PSCA inhibits the growth of the PSCA-expressing tumor cells (Figure 54). Exhibit 1 shows the results of mice injected with LAPC-9 cells and treated with mouse IgG, or 1G8 which is an IgG1 isotype, anti-PSCA monoclonal antibody. LAPC-9 cells are a human prostate cancer xenograft line which expresses high levels of PSCA. The mice represented in the upper panel were treated with a mouse IgG control, while the mice represented in the

lower panel were injected an anti-PSCA mAb cocktail. Tumor growth was monitored with caliper measurements. The control group included 6 mice (mice #1-6) and the 1G8 group included 7 mice (mice #7-13). The results show that the mice treated with the 1G8 antibody exhibited significant inhibition of tumor cell growth (Figure 54, lower panel) compared with the mice in the control group (Figure 54, upper panel). Thus, treatment with 1G8 alone inhibits the growth of tumor cells expressing PSCA.

7. Exhibits 2 and 3 depicts experimental results showing anti-PSCA monoclonal antibodies of different isotypes, each administered alone to animals that have been injected with tumor cells expressing PSCA, inhibit the growth of the PSCA-expressing tumor cells (Figure 55 A and B, respectively).

8. Exhibit 2 shows the results of mice injected with LAPC-9 and treated with mouse IgG, or 2A2, an IgG2a isotype, anti-PSCA monoclonal antibody (Figure 55A). The mice represented in Figure 55A were treated with either mouse IgG control (♦; diamonds), or with 2A2 (■; square). Tumor growth was monitored with caliper measurements. The results show that the mice treated with the 2A2 antibody exhibited significant inhibition of tumor cell growth (Figure 55A; ■) compared with the mice in the control group (Figure 55A; ♦). Tumor incidence was 6/6 mice in the mouse IgG control group, versus 2/7 for the 2A2-treated group. In the IgG control group, 4 out of the 6 mice had developed tumors by day 21. Thus, treatment with 2A2 alone inhibits the growth of tumor cells expressing PSCA.

9. Exhibit 3 shows the results of mice injected with LAPC-9 and treated with mouse IgG, or 2H9, an IgG1 isotype, anti-PSCA monoclonal antibody. The mice represented in Figure 55B were treated with either mouse IgG control (♦; diamonds), or with 2H9 (■; square). Tumor growth was monitored with caliper measurements. The control group included 6 mice and the 2H9 group included 7 mice. The results show that the mice treated with the

2H9 antibody exhibited significant inhibition of tumor cell growth compared with the mice in the control group. Tumor incidence was 6/6 mice in the mouse IgG control group, compared to 1/7 for the 2H9-treated group. In the 2H9-treated group the single tumor present appeared at day 21. In the mouse IgG control group, 4/6 of the mice had developed tumors by day 21. Thus, treatment with 2H9 alone inhibits the growth of tumor cells expressing PSCA.

10. Exhibit 4 depicts experimental results showing that administering an anti-PSCA antibody, to animals bearing established tumor cells, which express PSCA, inhibits the growth of the PSCA-expressing tumor cells (Figure 57). Exhibit 4 shows the results of mice bearing established LAPC-9 tumors (e.g., approximately 100 mm^3) treated with mouse IgG (♦; diamonds), or 3C5 (■; square) which is an IgG2a isotype, anti-PSCA monoclonal antibody. Tumor growth was monitored with caliper measurements. The results indicate that the 3C5 mAb inhibits the growth of established LAPC-9 prostate tumors. Some of the mice in the 3C5-treated group exhibited tumor regression up to 50% of the initial, pre-treatment size. Thus, treatment with 3C5 alone inhibits the growth of established tumor cells expressing PSCA.

11. Exhibit 5 depicts experimental results showing that the PSCA monoclonal antibodies exert tumor growth inhibition specifically through the PSCA protein on a PSCA-expressing cell (Figure 65). Exhibit 5 shows the results of mice injected with LAPC-9 cells which express PSCA (upper panel, Figure 65), or PC-3 which do not express PSCA (lower panel, Figure 65), and treated with mouse IgG or 1G8. Tumor growth was monitored with caliper measurements. The results show that the mice bearing LAPC-9 (Figure 65, upper panel; ♦) or PC-3 tumors (Figure 65, lower panel; ♦) and treated with mouse IgG exhibited significant tumor growth over a 40-day period. The mice bearing PC-3 tumors and treated with 1G8 also exhibited significant tumor growth (Figure 64, lower panel; ■). In contrast, the mice bearing LAPC-9 tumors and treated with 1G8

exhibited inhibited tumor growth. Thus, anti-PSCA antibodies, such as 1G8, inhibit tumor growth specifically through the PSCA protein on a PSCA-expressing cell.

12. Exhibit 6 depicts experimental results showing that administering an anti-PSCA antibody to animals bearing established orthotopic tumor cells which express PSCA inhibits the level of increase of serum PSA and inhibits the growth of the PSCA-expressing tumor cells (Figure 66A and B). The serum level of PSA was used to track the growth of the tumors, since the serum PSA level correlated well with the tumor size. The mice were segregated into two treatment groups, based on the levels of serum PSA. The group having low levels of PSA is depicted in Figure 66A and the group having moderate levels of PSA is depicted in Figure 66B. Exhibit 6 shows the results of mice bearing established orthotopic LAPC-9 tumors treated with phosphate buffer saline (PBS; ◆; diamonds), or 1G8 (■; square) which is an IgG1 isotype, anti-PSCA monoclonal antibody. The mice treated with 1G8, exhibited a reduction in the rate of increase in serum PSA levels (Figure 66A and B), compared with the mice treated with PBS. Thus, treatment with 1G8 alone reduces the rate of increase in serum PSA levels, which correlates with inhibiting the growth of tumors expressing PSCA.

13. Exhibit 7 depicts experimental results showing that administering an anti-PSCA antibody, to animals bearing established orthotopic tumor cells which express PSCA, increased the survival of the tumor-bearing mice (Figure 67A and B). The mice described in Exhibit 7 (e.g., treated with PBS or 1G8) were permitted to live, to determine the length of survival time. The mice treated with PBS began to die within 5-6 weeks post-injection, due to local tumor growth and metastasis. In contrast, the mice treated with 1G8 antibody exhibited a prolonged life. The mice having lower serum PSA levels and treated with 1G8 (Figure 67A) exhibited the greatest increase in survival. The inhibition of tumor growth correlated with prolonged life. Thus, treatment with 1G8 increased survival time, by inhibiting tumor growth.

14. Exhibit 8 depicts experimental results showing that administering an anti-PSCA antibody to animals bearing established orthotopic tumor cells which express PSCA inhibits the level of increase of serum PSA and inhibits the growth of the PSCA-expressing tumor cells (Figure 68A and B). The serum level of PSA was used to track the growth of the tumors, since the serum PSA level correlated well with the tumor size. The mice were segregated into two treatment groups, based on the levels of serum PSA. The group having low levels of PSA is depicted in Figure 68A and the group having moderate levels of PSA is depicted in Figure 68B. Exhibit 8 shows the results of mice bearing established orthotopic LAPC-9 tumors treated with phosphate buffer saline (PBS; ♦; diamonds), or 3C5 (■; square) which is an IgG2a isotype, anti-PSCA monoclonal antibody. The mice treated with 3C5, exhibited a reduction in the rate of increase in serum PSA levels (Figure 68A and B), compared with the mice treated with PBS. Thus, treatment with 3C5 alone reduces the rate of increase in serum PSA levels, which correlates with inhibits growth of tumors expressing PSCA.

15. Exhibit 9 depicts experimental results showing that administering an anti-PSCA antibody, to animals bearing established orthotopic tumor cells which express PSCA, increased the survival of the tumor-bearing mice (Figure 69A and B). The mice described in Exhibit 9 (e.g., treated with PBS or 3C5) were permitted to live, to determine the length of survival time. The mice treated with PBS began to die within 5-6 weeks post-injection, due to local tumor growth and metastasis. In contrast, the mice treated with 3C5 antibody exhibited a prolonged life. The mice having lower serum PSA levels and treated with 3C5 (Figure 69A) exhibited the greatest increase in survival. The inhibition of tumor growth correlated with prolonged life. Thus, treatment with 3C5 increased survival time, by inhibiting tumor growth.

16. Exhibit 10 depicts experimental results showing that administering an anti-PSCA antibody alone or in combination with doxorubicin to animals bearing established tumor cells which express PSCA inhibits the growth of the PSCA-expressing tumor cells (Figure 70). Exhibit 10 shows the results of mice bearing established PC3-PSCA tumors (e.g., approximately 100-200 mm^3) treated with PBS, 1G8 alone, doxorubicin alone, or a combination of 1G8 and doxorubicin. The PC3-PSCA cells were derived by retroviral gene transfer of the PSCA gene into PC3 cells which do not express PSCA and are androgen-independent. Tumor growth was monitored with caliper measurements. The mice treated with doxorubicin alone exhibited a slightly lower tumor growth rate, compared to mice treated with PBS. In contrast, mice treated with 1G8 antibody alone exhibited a greater reduction in tumor growth rate, compared to the mice treated with PBS. The mice treated with the combination of 1G8 and doxorubicin exhibited a slightly greater reduction in tumor growth rate, compared to the mice treated with PBS, 1G8 alone or doxorubicin alone. Thus, treatment with 1G8 in combination with doxorubicin inhibits the growth of established, androgen-independent tumor cells expressing PSCA.

17. Exhibit 11 depicts experimental results showing that an anti-PSCA monoclonal antibody, administered to a tumor-bearing animal, selectively targets tumor cells expressing PSCA (Figure 71). Exhibit 11 shows the results of immunohistochemistry analyses of tumor explants from the mice described in Exhibit 4. These mice bear established LAPC-9 tumors (e.g., approximately 100 mm^3) and were treated with mouse IgG, or 3C5 which is an IgG2a isotype, anti-PSCA monoclonal antibody. The tumor explants from the mice in both treated groups were analyzed by immunohistochemistry. The tumor explants were sliced and fixed, and probed with goat anti-mouse IgG to detect the presence of antibody. The results show that the 3C5 antibody was detected throughout the tumor explants and was localized specifically in the tumor explants expressing PSCA but not in the cells surrounding the tumor. In contrast, no antibody was detected in the tumor explants from

the mice treated with IgG. Thus, the administered 3C5 monoclonal antibody specifically localized to tumor cells expressing PSCA.

18. Exhibit 12 depicts experimental results showing that an anti-PSCA monoclonal antibody, administered to a tumor-bearing animal, selectively targets tumor cells expressing PSCA (Figure 72). Exhibit 12 shows the results of a Western blot analysis of tumor lysates from the mice described in Exhibit 4. These mice bear established LAPC-9 tumors (e.g., approximately 100 mm^3) and were treated with mouse IgG, or 3C5 which is an IgG2a isotype, anti-PSCA monoclonal antibody. The tumor lysates from the mice in both treated groups were analyzed by Western blot analysis. The tumor lysates were electrophoresed in a gel, transferred to a solid support, and the solid support was probed with goat anti-mouse IgG-HRP antibodies to detect the presence of antibody. The mouse IgG control antibody and 3C5 were also run on the gel as controls. The results show that the that IgG heavy and light chains were readily detected in tumor lysates from the 3C5-treated mice, but not in the mouse IgG control treated mice. Thus, the administered 3C5 monoclonal antibody specifically localized to tumor cells expressing PSCA.

19. Exhibit 13 depicts experimental results showing that an anti-PSCA monoclonal antibody, administered to a tumor-bearing animal, selectively targets tumor cells expressing PSCA (Figure 73). Exhibit 13 shows the results of a Western blot analysis of tumor lysates from mice treated with 1G8 or mouse IgG. These mice bear established LAPC-9 tumors (e.g., approximately 100 mm^3) and were treated with mouse IgG, or 1G8 which is an IgG1 isotype, anti-PSCA monoclonal antibody. The tumor lysates from the mice in both treated groups were analyzed by Western blot analysis. The tumor lysates were electrophoresed in a gel, transferred to a solid support, and the solid support was probed with goat anti-mouse IgG-HRP antibodies to detect the presence of antibody. The mouse IgG control antibody and 1G8 were also run on the gel as controls. The results show that the that IgG heavy and light chains were readily detected in tumor lysates from the 1G8-

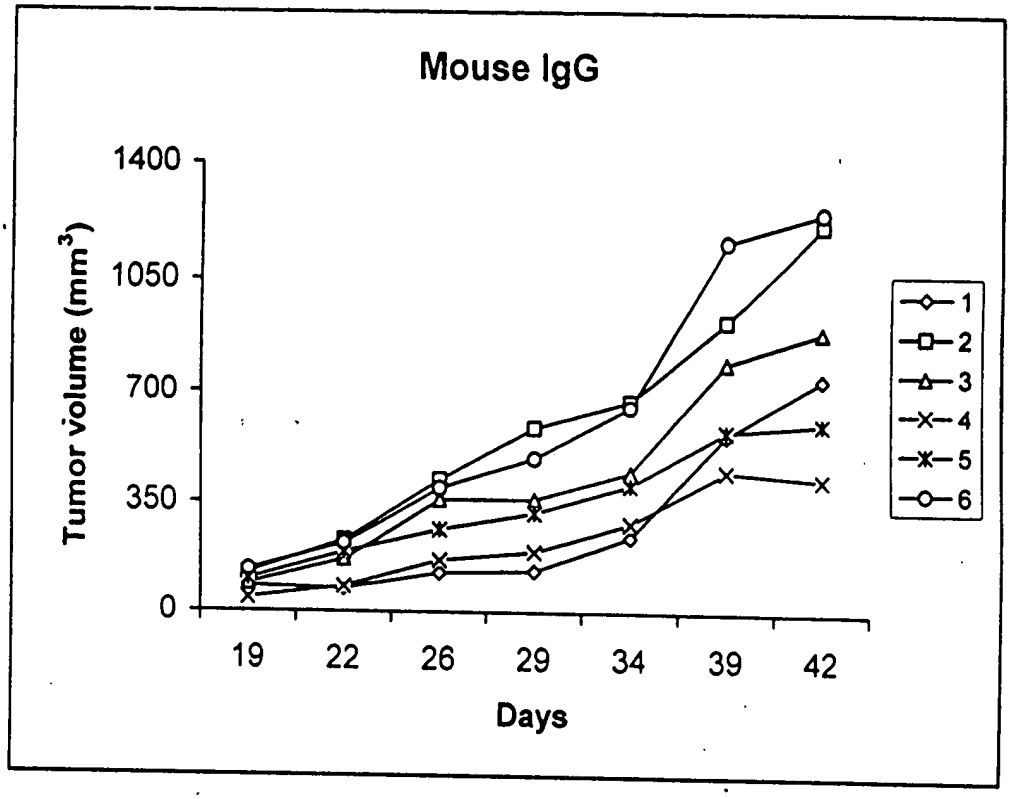
treated mice, but not in the mouse IgG control treated mice. Thus, the administered 1G8 monoclonal antibody specifically localized to tumor cells expressing PSCA.

We further declare that all statements made herein of our own knowledge are true and that all statements made on information and belief are believed to be true and further that these statements were made with the knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States Code and that willful false statements may jeopardize the validity of the application or any patent issuing thereon.

DATE	Robert E. Reiter
Aug 30, 2000	Owen Witte
DATE	Owen N. Witte

EXHIBIT 1

FIG. 54


Mouse IgG
Tumor volume (mm3)
1400
1050
700
350
0
19
22
26
29
34
39
42
Days
1
2
3
4
5
6

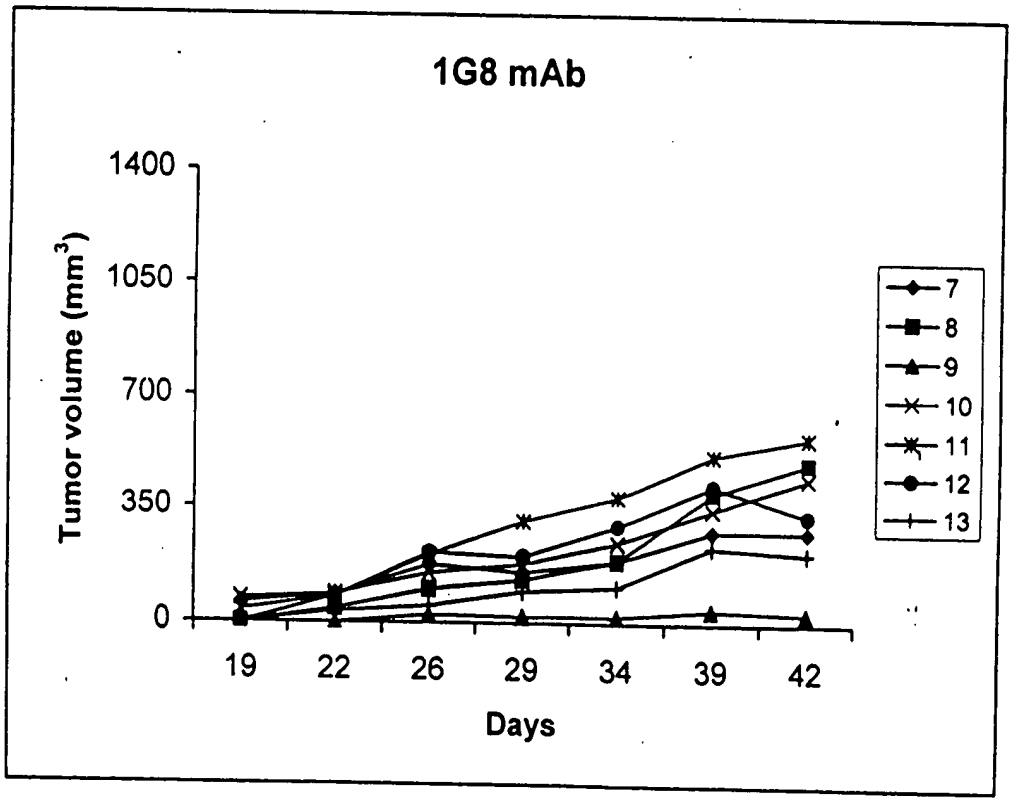

1G8 mAb
Tumor volume (mm3)
1400
1050
700
350
0
19
22
26
29
34
39
42
Days
7
8
9
10
11
12
13

EXHIBIT 2

FIG. 55 A

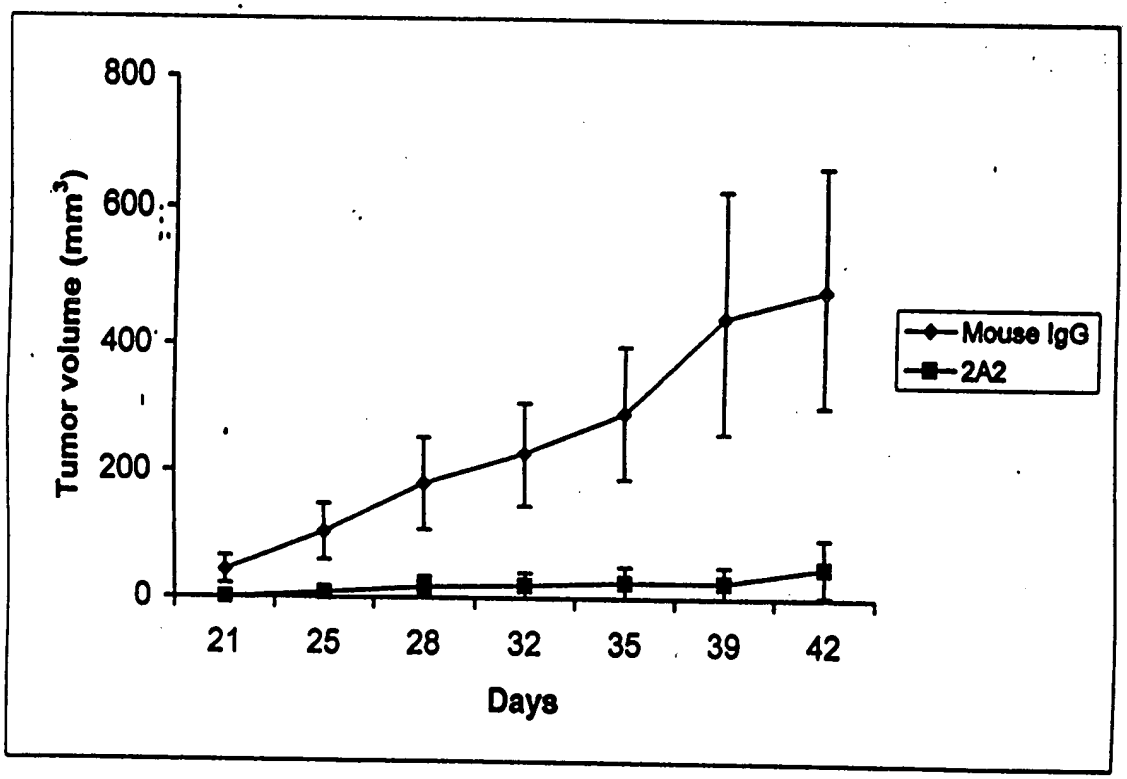

800
600
400
200
0
Tumor volume (mm3)
21
25
28
32
35
39
42
Days
Mouse IgG
2A2

EXHIBIT 3

FIG. 55 B

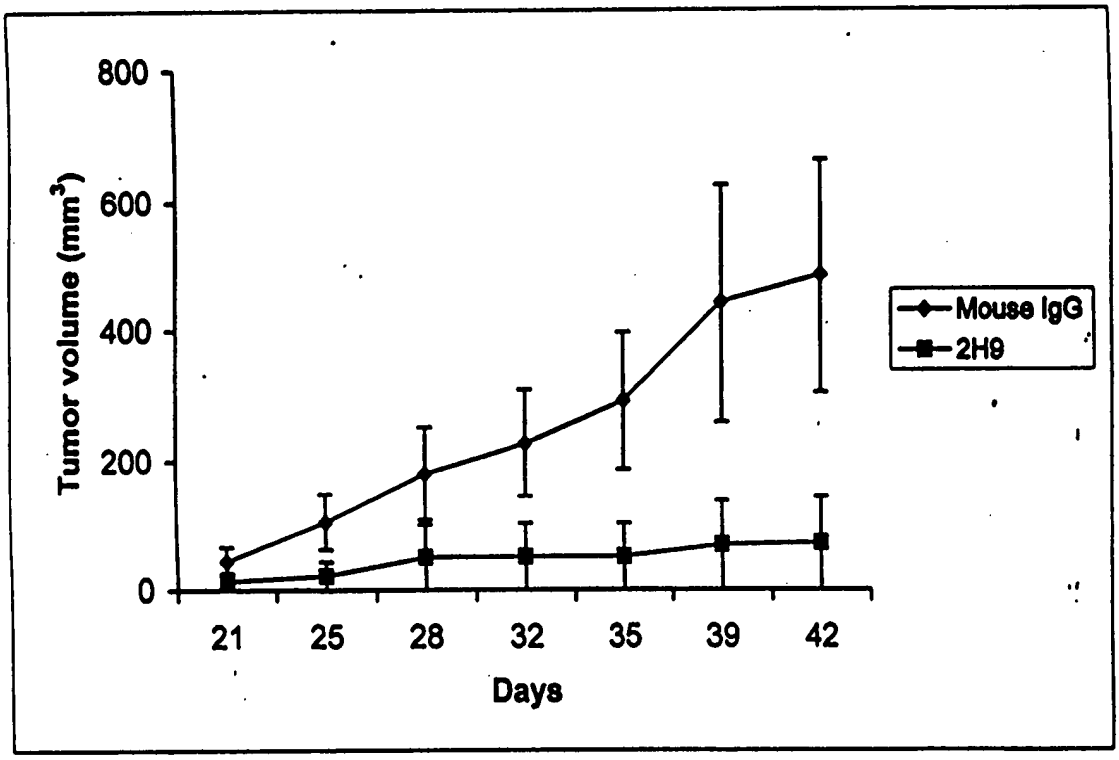

800
600
400
200
0
Tumor volume (mm3)
21
25
28
32
35
39
42
Days
Mouse IgG
2H9

EXHIBIT 4

FIG. 57

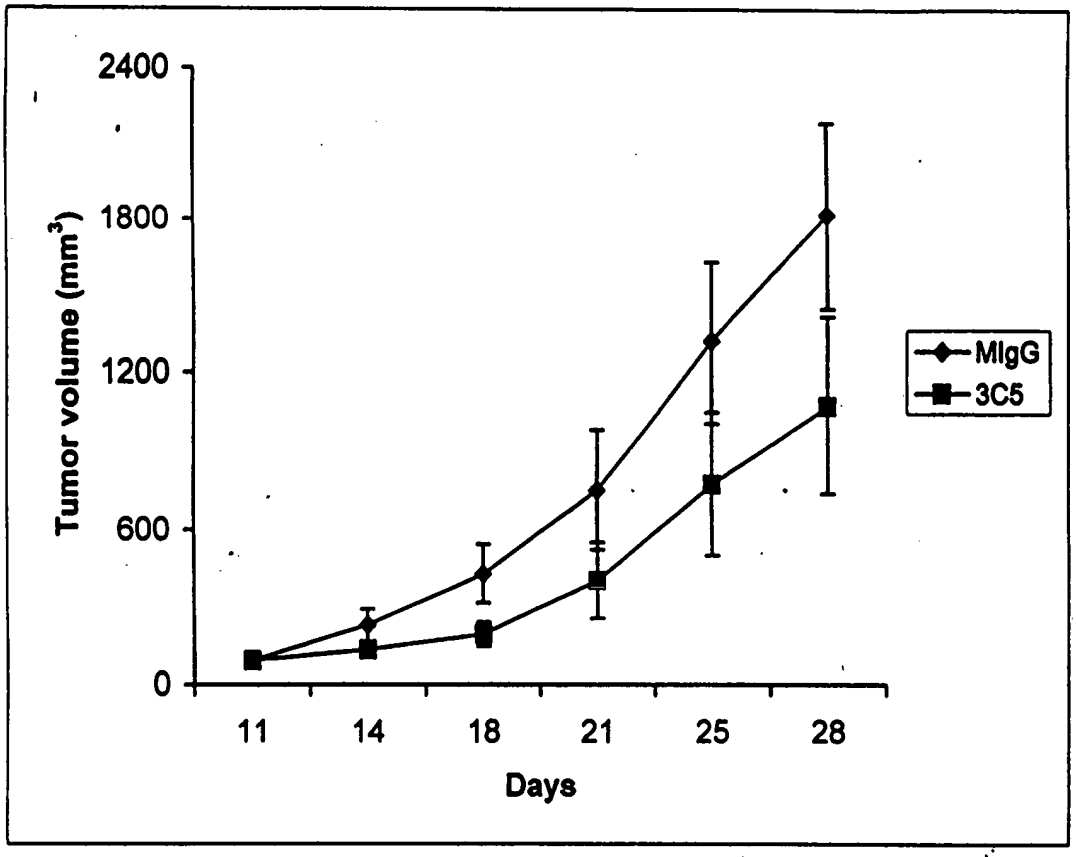
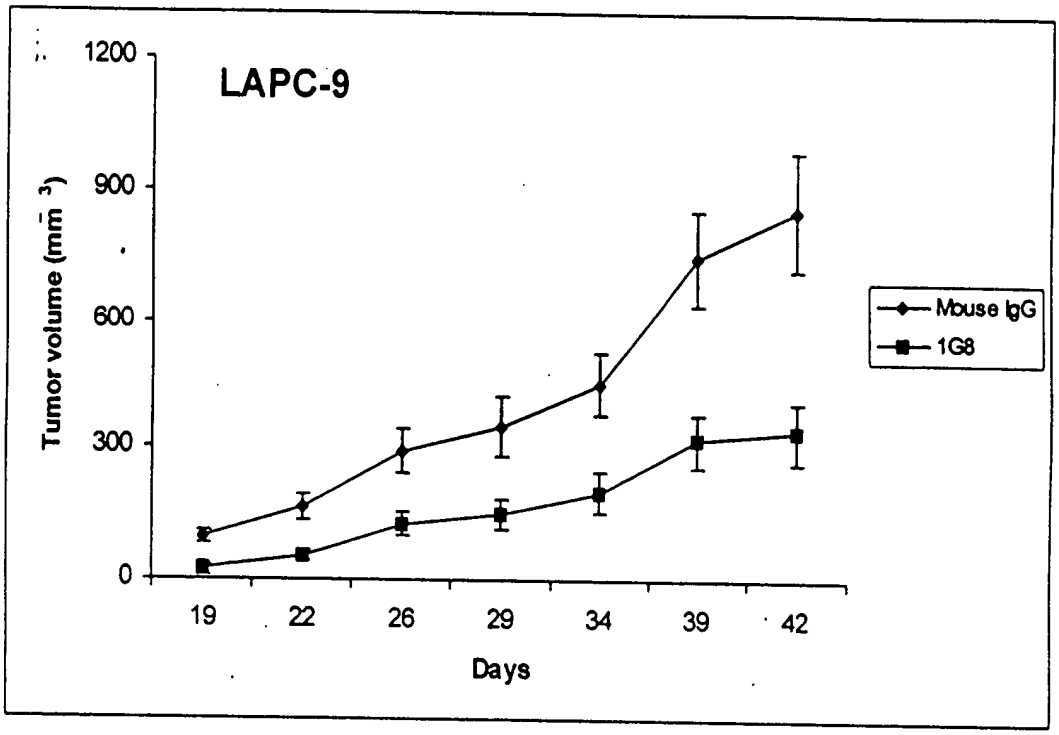

2400
1800
1200
600
0
Tumor volume (mm3)
11
14
18
21
25
28
Days
MIgG
3C5

EXHIBIT 5


LAPC-9
Tumor volume (mm 3)
1200
900
600
300
0
19
22
26
29
34
39
42
Days
Mouse IgG
1G8

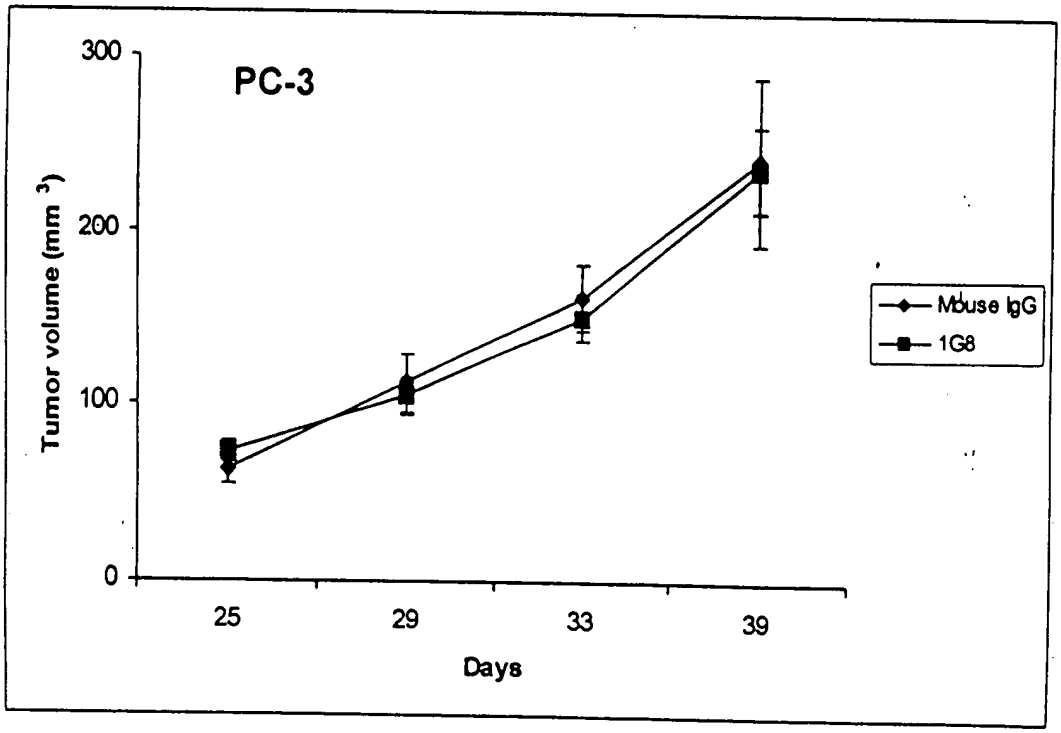
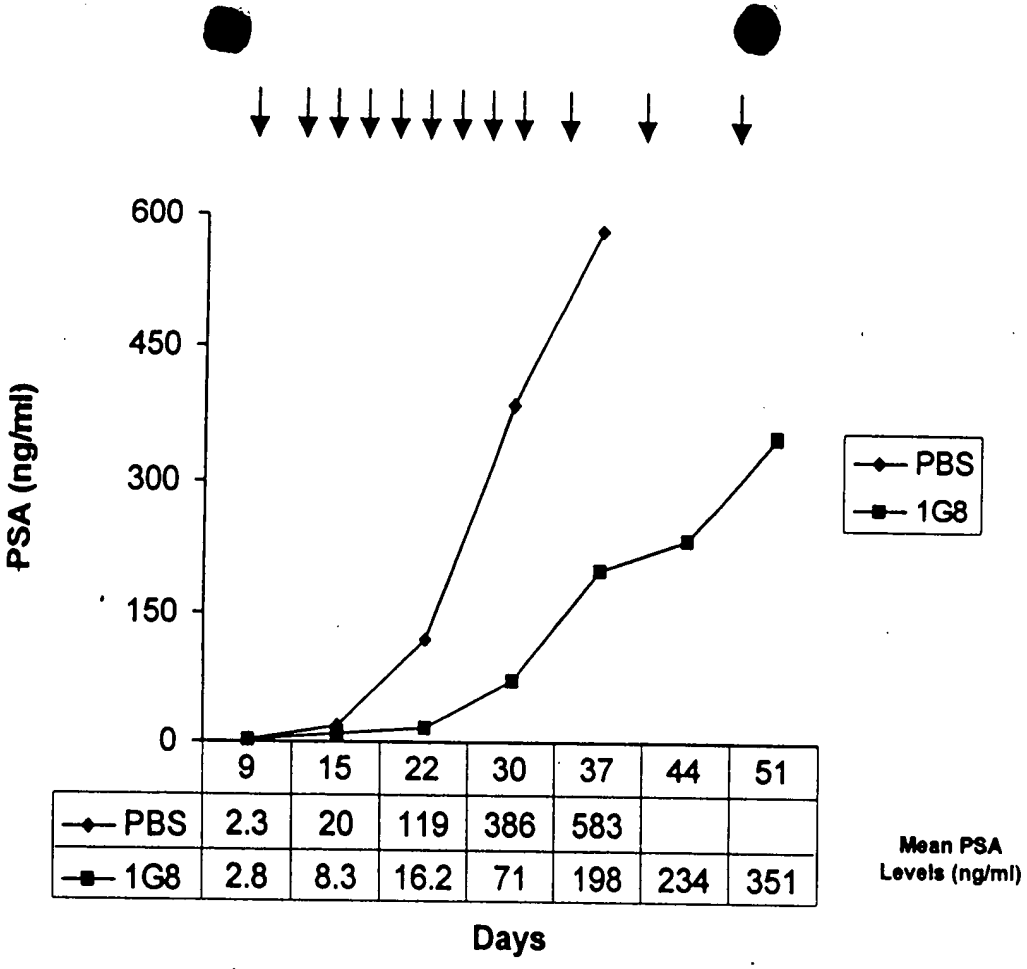

PC-3
Tumor volume (mm 3)
300
200
100
0
25
29
33
39
Days
Mouse IgG
1G8

FIGURE 65

EXHIBIT 6

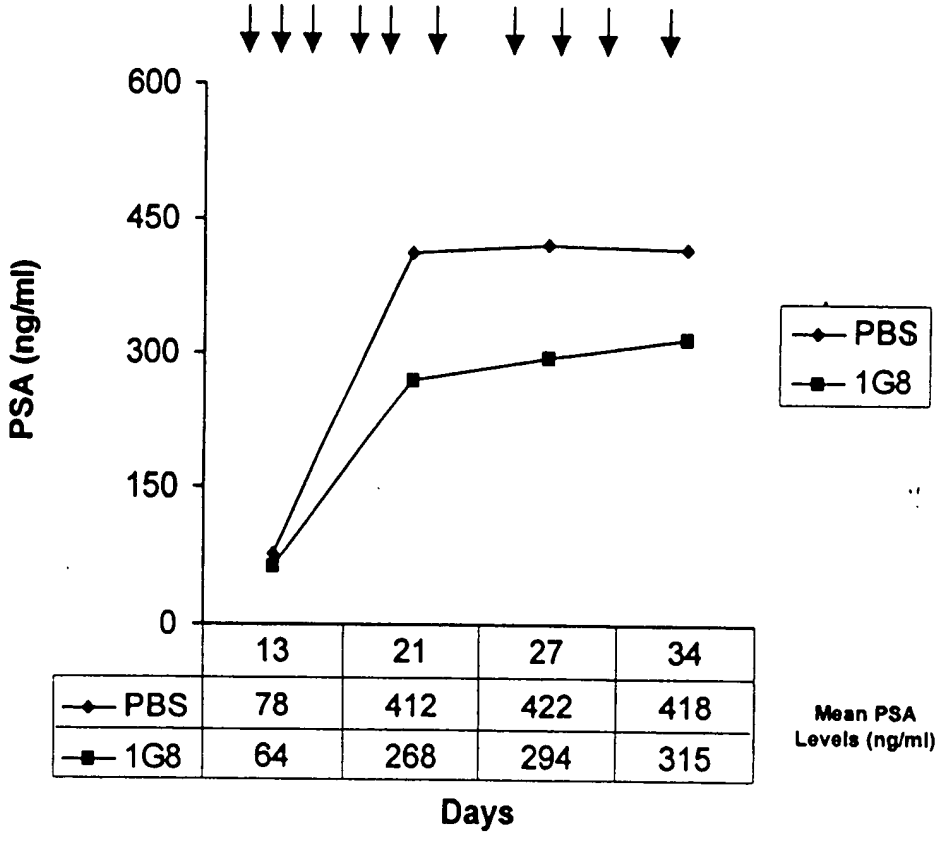
A)



	9	15	22	30	37	44	51
PBS	2.3	20	119	386	583		
1G8	2.8	8.3	16.2	71	198	234	351

Mean PSA Levels (ng/ml)

B)



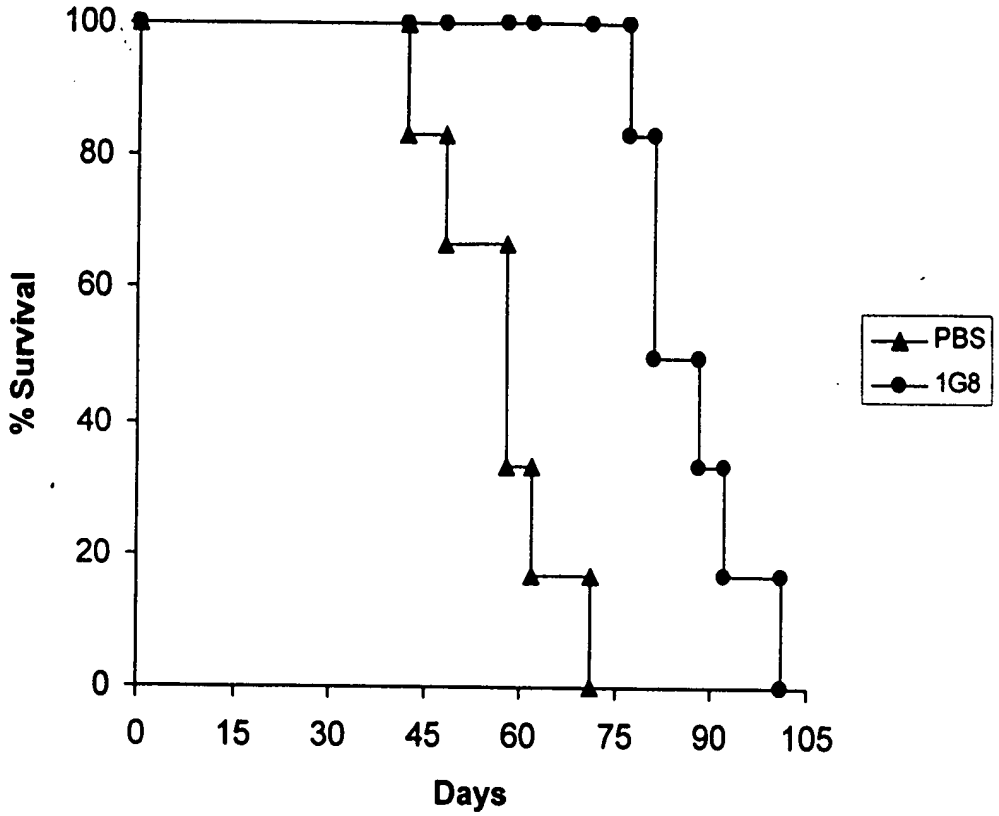
	13	21	27	34
PBS	78	412	422	418
1G8	64	268	294	315

Mean PSA Levels (ng/ml)

Figure 66

EXHIBIT 7

A)


100
80
60
40
20
0
% Survival
0
15
30
45
60
75
90
105
Days
PBS
1G8

B)

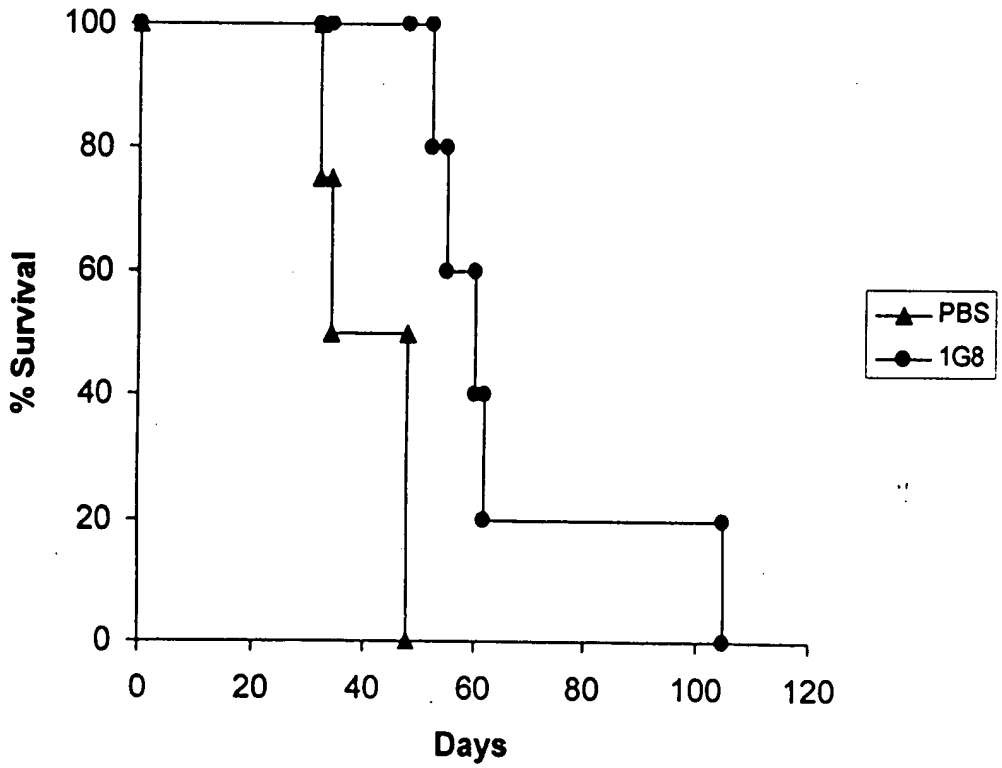
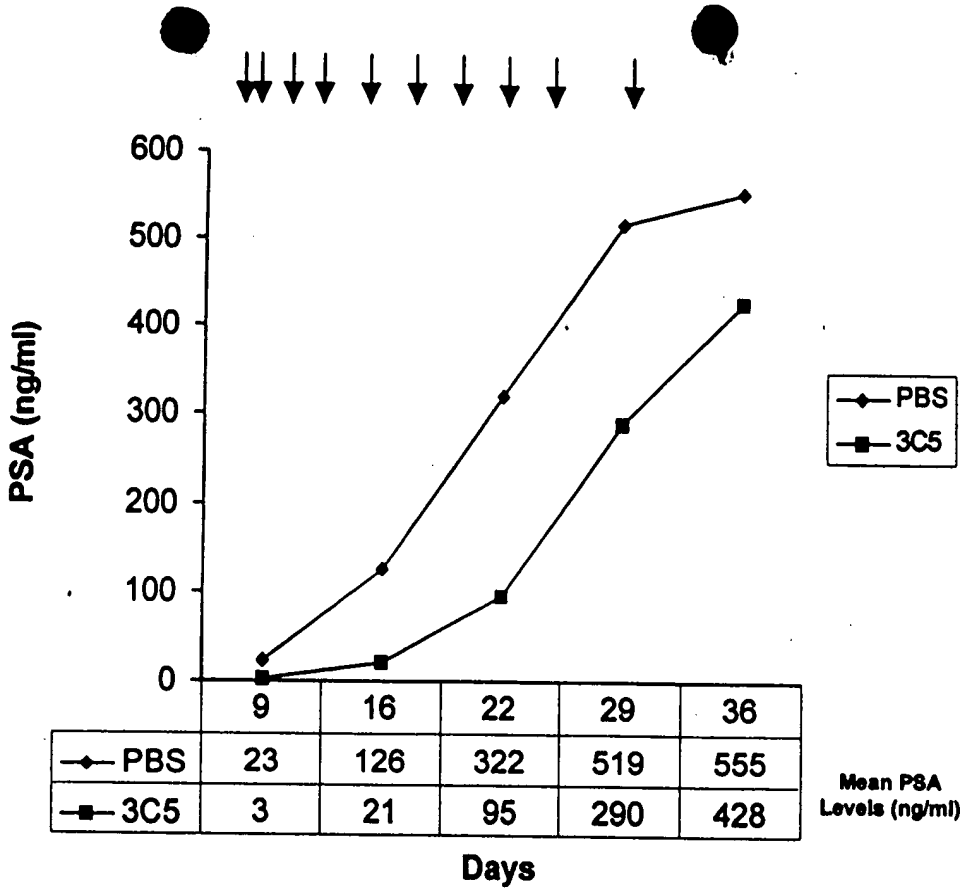

100
80
60
40
20
0
% Survival
0
20
40
60
80
100
120
Days
PBS
1G8

Figure 67

EXHIBIT 8

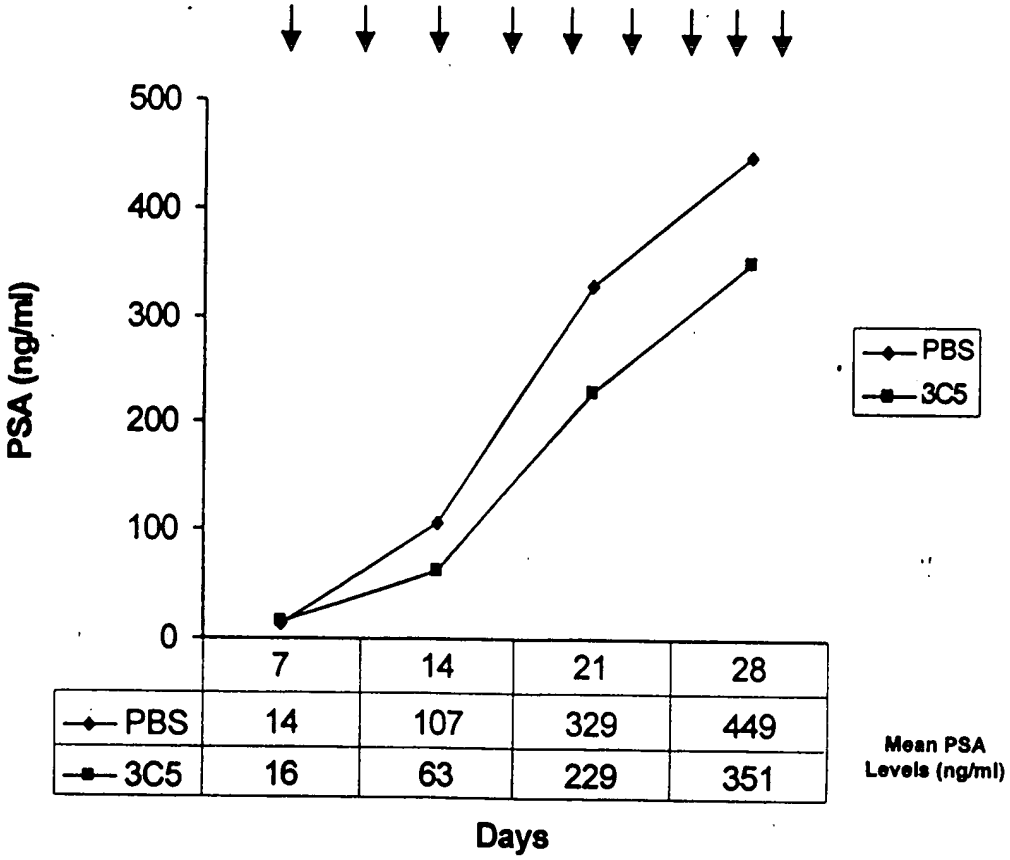
A)



	9	16	22	29	36
PBS	23	126	322	519	555
3C5	3	21	95	290	428

Mean PSA Levels (ng/ml)

B)



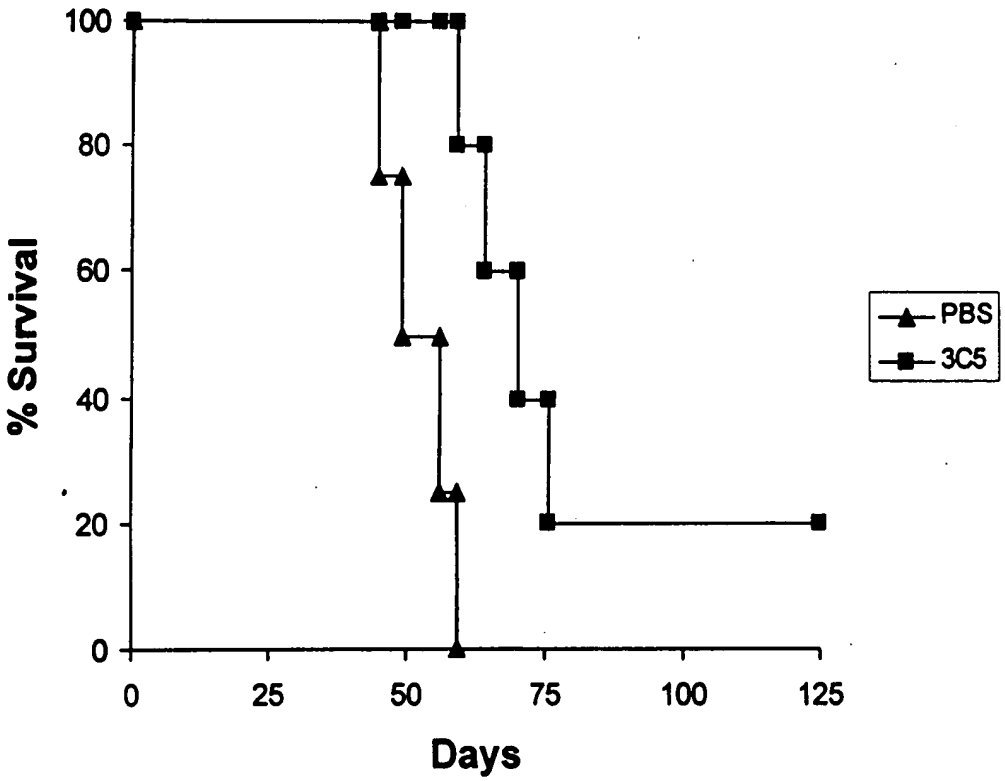
	7	14	21	28
PBS	14	107	329	449
3C5	16	63	229	351

Mean PSA Levels (ng/ml)

Figure 68

EXHIBIT 9

A)


100
80
60
40
20
0
% Survival
0
25
50
75
100
125
Days
PBS
3C5

B)

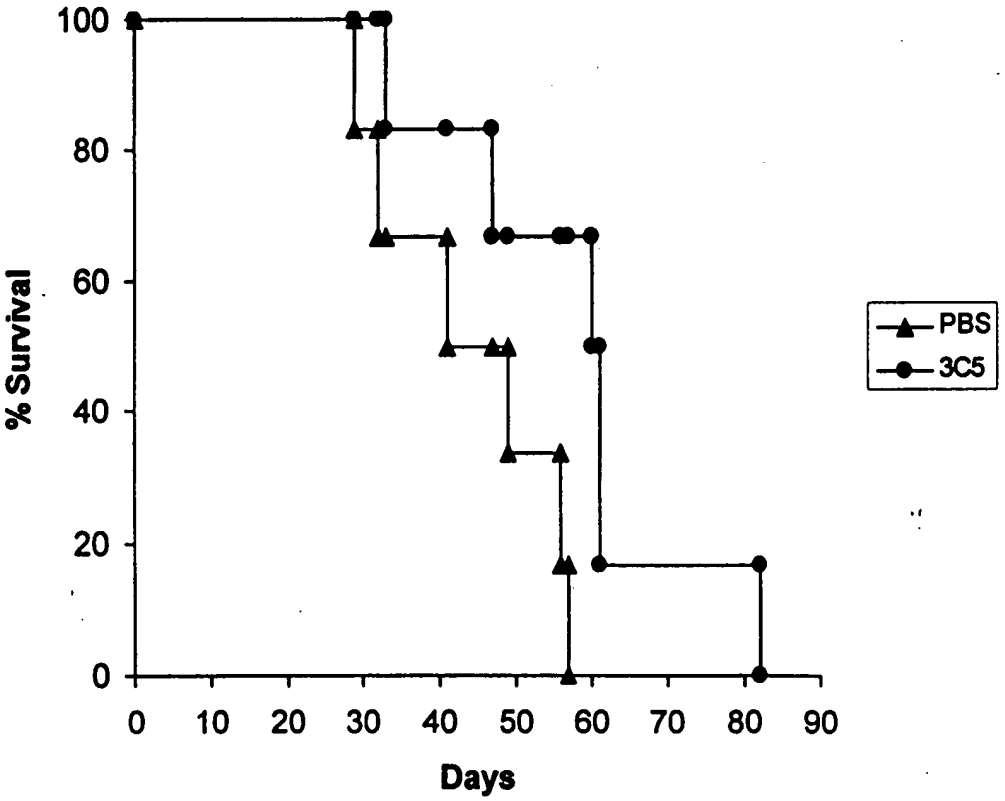
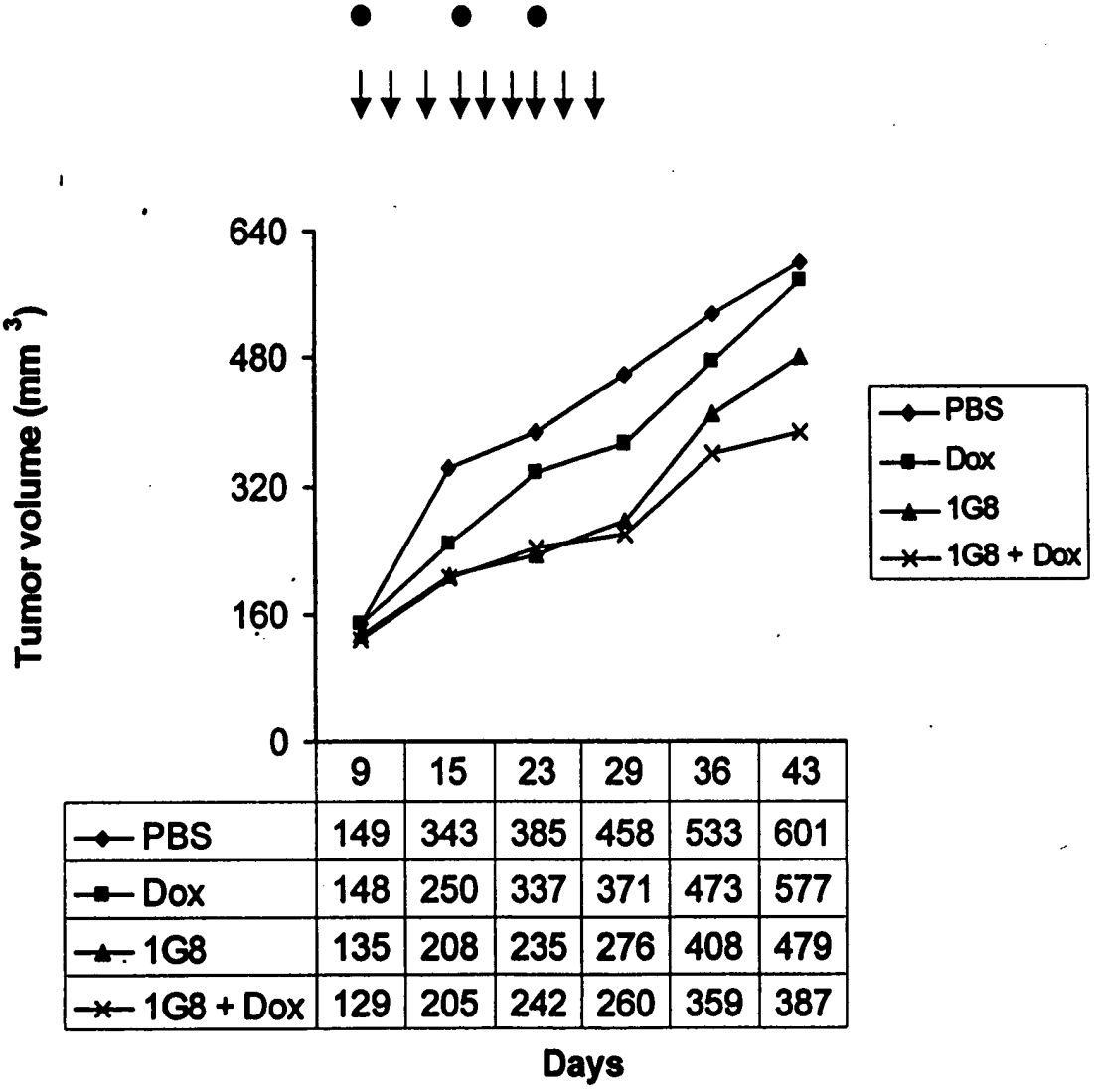

100
80
60
40
20
0
% Survival
0
10
20
30
40
50
60
70
80
90
Days
PBS
3C5

Figure 69

EXHIBIT 10



	9	15	23	29	36	43
PBS	149	343	385	458	533	601
Dox	148	250	337	371	473	577
1G8	135	208	235	276	408	479
1G8 + Dox	129	205	242	260	359	387

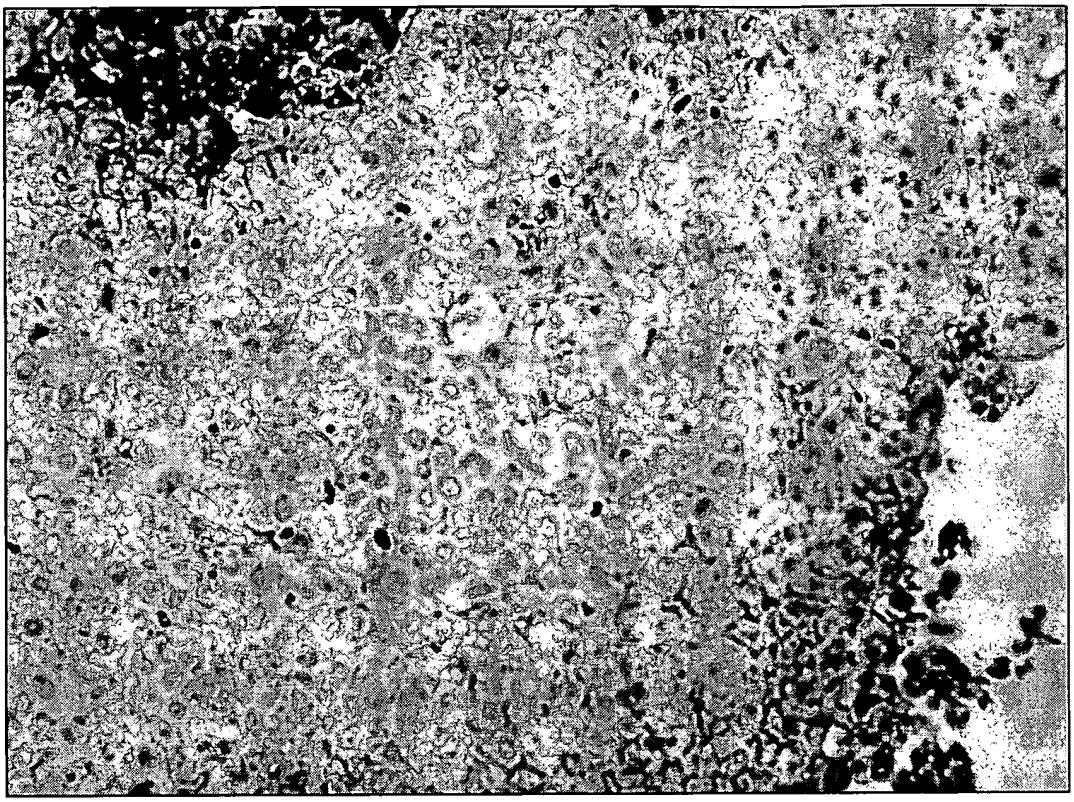
Days

Figure 70

EXHIBIT 11

PSCA 3C5 MAb Localizes within LAPC9AD Xenograft Tissue

3C5 Treated



mIgG Treated

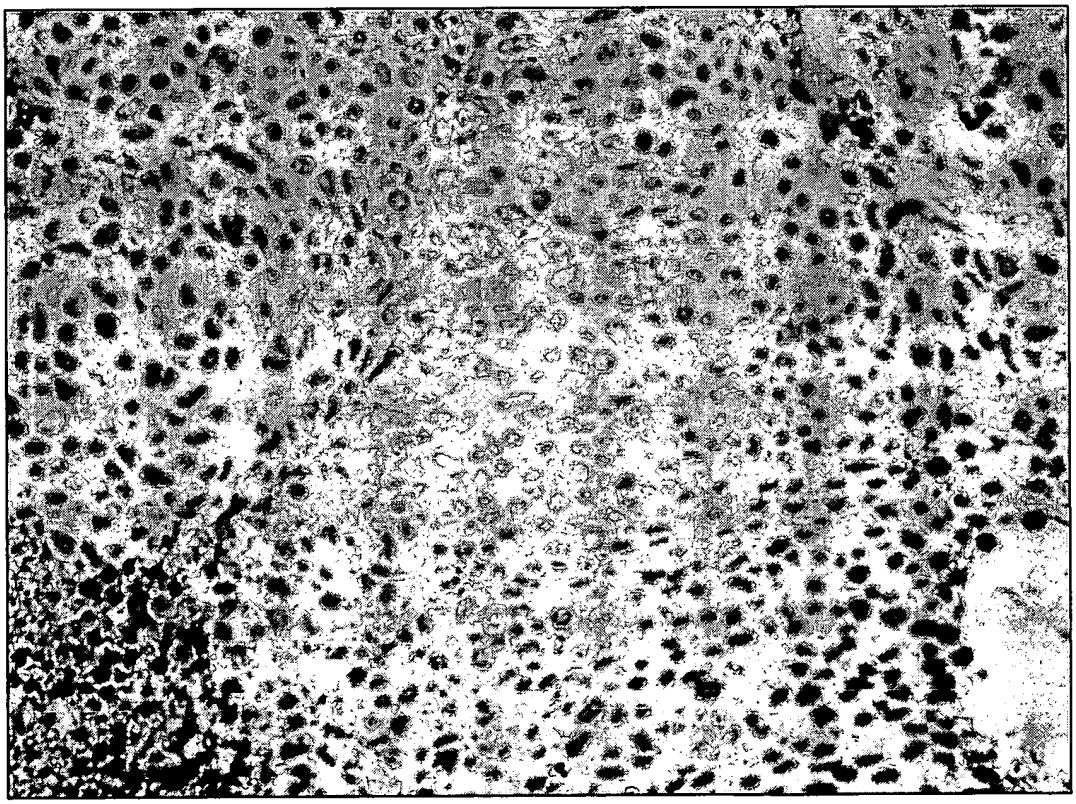
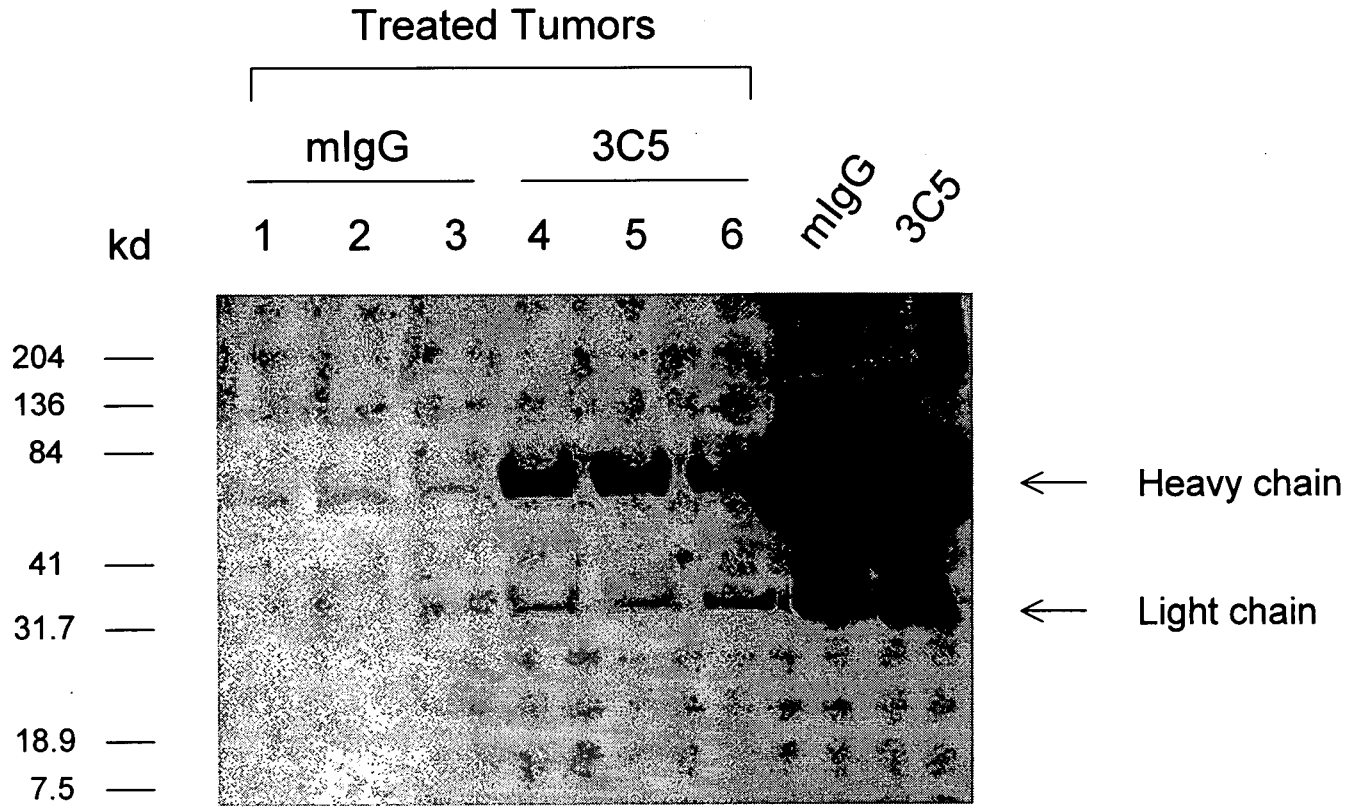


Figure 71

EXHIBIT 12

3C5 Anti-PSCA MAb is Localized to Established LAPC-9 Tumors

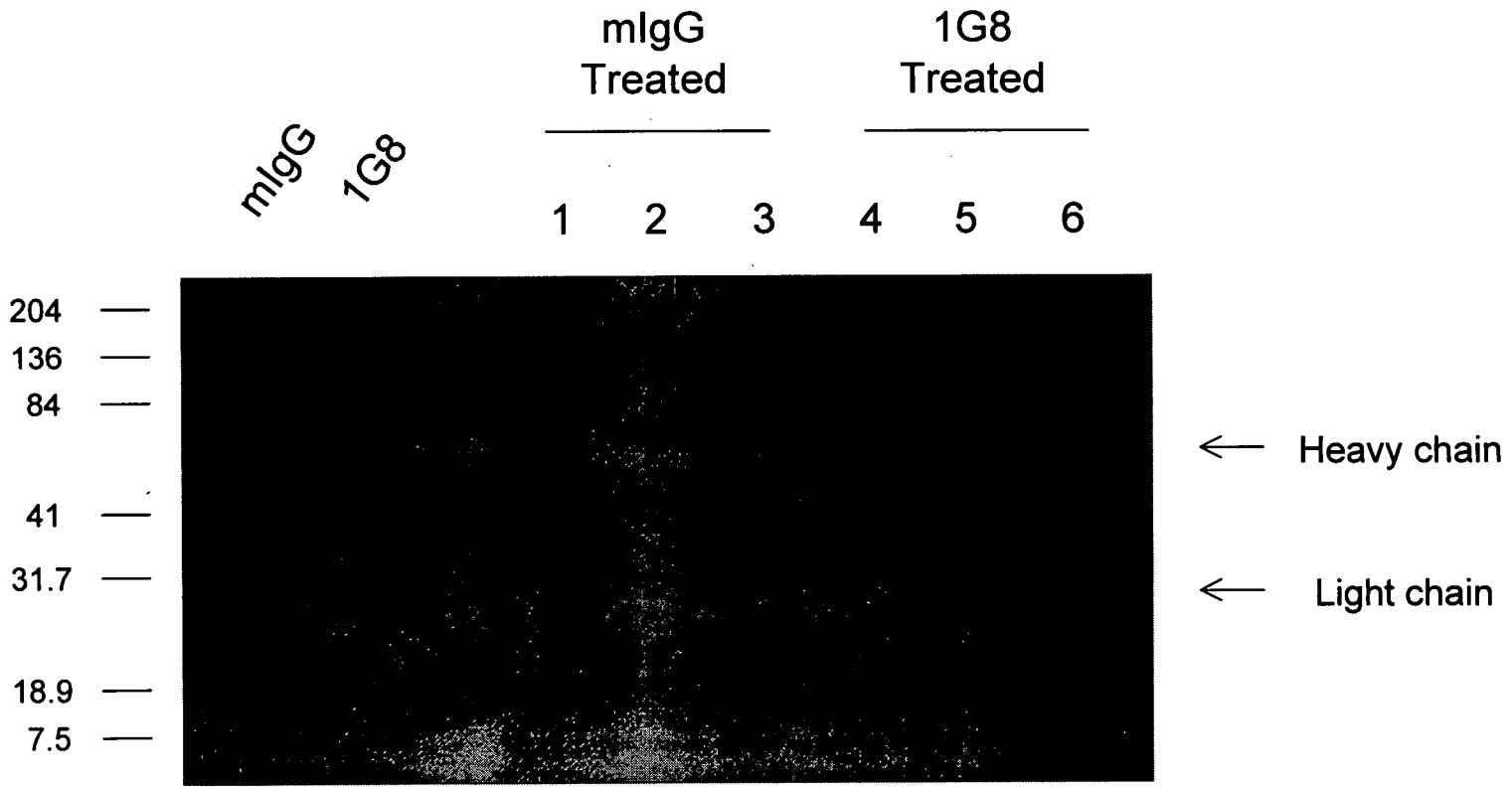


Western blot developed with α-mIgG/κ

Figure 72

EXHIBIT 13

SPECIFIC TARGETING OF THE 1G8 ANTI-PSCA MAb TO ESTABLISHED LAPC-9 TUMORS



- α-MIgG Western

Method: Mice bearing established LAPC-9 tumors (>100 mm^3) were injected with either mIgG or the anti-PSCA MAb 1G8. Tumors were harvested a week later and made into protein lysates for Western analysis.

Figure 73

MANDEL & ADRIANO

INTELLECTUAL PROPERTY ATTORNEYS

35 N. ARROYO PARKWAY, SUITE 60
PASADENA, CALIFORNIA 91103
PHONE 626/395-7801
FAX 626/395-0694

ATTACH TO #17½ LCH

FACSIMILE TRANSMISSION

To: Examiner Larry Helms
Company, Firm, or Institution: US Patent and Trademark Office
Facsimile No.: 703-308-4315
Total No. of pages (including this page): 6

From: Roberta German, patent agent
Date: October 5, 2000
U.S. Serial No.: 08/814,279
Our Ref.: 30436.54 US01

Dear Larry,

I am FAXing you a copy of the granted petition (Exhibit 1), which grants treating US Serial No. 08/814,279 as a provisional application having the filing date of March 10, 1997. I hope the granted petition will be helpful in clarifying this matter. For your convenience, I have included a second page which tracks the relationship of the various applications. Please contact me if you have any questions.

Sincerely,
Roberta German

RELATIONSHIP OF THE PATENT APPLICATIONS:

US Serial No:	Filing date:	M & A Ref. No.:	Relationship:
08/814,279	Mar 10, 1997	30435.54 US01	Filed as a non-provisional appl'n, converted to a provisional, and then abandoned in favor of 09/038,261. See granted petition attached as Exhibit 1.
60/071,141	Jan 12, 1998	30435.54 USP2	Filed as a provisional appl'n, and then abandoned in favor of 09/038,261.
60/074,675	Feb 13, 1998	30435.54 USP3	Filed as a provisional appl'n, and then abandoned in favor of 09/038,261.
09/038,261	Mar 10, 1998	30435.54 USU1	Filed as a non-provisional appl'n, claiming priority of 08/814,279, 60/071,141, and 60/074,675.

EXHIBIT 1

UNITED STATES PATENT AND TRADEMARK OFFICE

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ROBERT A. MILLMAN
MORRISON & FOERSTER
2000 PENNSYLVANIA AVE., N.W.
WASHINGTON, DC 20006-1888

In re Application of
Reiter
Application No. 08/814,279
Filed: 10 March, 1997
Attorney Docket No. 220002057500

DECISION ON PETITION

This is a decision on the petition filed (with fee) on 14 June, 1997 (the June 1997 petition), and resubmitted on 19 January, 1999 (the January 1999 petition).

The Office regrets the delay in addressing this matter.

The petition requests that the Nonprovisional Application filed 10 March, 1997, be treated as a Provision application under 37 C.F.R. §1.53(c), (g), and (i),[1] rather than under 37 C.F.R. 1.53(b),[2] and be accorded a filing date of 10 March, 1997. Unspoken

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[1] The regulations at 37 C.F.R. §1.53(d), (g), and (i) provide:

§1.53 Application number, filing date, and completion of application.

...

(d) Application filing requirements - Provisional application. The filing date of a provisional application is the date on which a specification as prescribed by the first paragraph of 35 U.S.C. 112, and any drawing required by § 1.81(a) are filed in the Patent and Trademark Office. No amendment, other than to make the provisional application comply with the patent statute and all applicable regulations, may be made to the provisional application after the filing date of the provisional application.

(1) A provisional application must also include the cover sheet required by § 1.51(c)(1) or a cover letter identifying the application as a provisional application. Otherwise, the application will be treated as an application filed under paragraph (b) of this section.

(2) An application for patent filed under paragraph (b) of this section may be converted to a provisional application and be accorded the original filing date of the application filed under paragraph (b) of this section.

(i) Provided that a petition requesting the conversion, with the fee set forth in § 1.17(q), is filed prior to the earliest of:

(A) Abandonment of the application filed under paragraph (b) of this section;

(B) Payment of the issue fee on the application filed under paragraph (b) of this section;

(C) Expiration of twelve months after the filing date of the application filed under paragraph (b) of this section; or

(D) The filing of a request for a statutory invention registration under § 1.293 in the application filed under paragraph (b) of this section.

(ii) The grant of any such petition will not entitle applicant to a refund of the fees which were properly paid in the application filed under paragraph (b) of this section.

(3) A provisional application is not entitled to the right of priority under 35 U.S.C. 119 or 365(a) or § 1.55, or to the benefit of an earlier filing date under 35 U.S.C. 120, 121 or 365(c) or § 1.78 of any other application. No claim for priority under § 1.78(a)(3) may be made in a design application based on a provisional application. No request under § 1.293 for a statutory invention registration may be filed in a provisional application. The requirements of §§ 1.821 through 1.825 regarding application disclosures containing nucleotide and/or amino acid sequences are not mandatory for provisional applications.

...

(g) Completion of application subsequent to filing -Provisional application. If a provisional application which has been accorded a filing date pursuant to paragraph (c) of this section does not include the appropriate filing fee or the cover sheet required by § 1.51(c)(1), applicant will be so notified, if a correspondence address has been provided, and given a period of time within which to file the fee, cover sheet, and the surcharge as set forth in § 1.16(f) in order to prevent abandonment of the application. If the required filing fee is not timely paid, the application may be disposed of. The notification pursuant to this paragraph may be made simultaneously with any notification pursuant to paragraph (e) of this section. If no correspondence address is included in the application, applicant has two months from the filing date to file the basic filing fee, cover sheet and the surcharge as set forth in § 1.16(f) in order to prevent abandonment of the application.

...

(i) Subsequent treatment of application - Provisional application. A provisional application for a patent filed under paragraph (c) of this section will not be placed on the files for examination and will become abandoned no later than twelve months after its filing date pursuant to 35 U.S.C. 111(b)(1).

...

[48 Fed. Reg. 2709, Jan. 20, 1983, effective Feb. 27, 1983; paras. (b) and (d), 48 Fed. Reg. 554, Jan. 4, 1984, effective Apr. 1, 1984; para. (c), 60 Fed. Reg. 31828, Aug. 6, 1995, effective Oct. 6, 1995; paras.(c) and (s), 53 Fed. Reg. 47808, Nov. 28, 1988, effective Jan. 1, 1989; paras. (b) and (c), 54 Fed. Reg. 47518, Nov. 15, 1989, effective Jan. 18, 1990; paras. (a)-(e) revised, 60 Fed. Reg. 20195, Apr. 25, 1995, effective June 8, 1995; revised, 62 Fed. Reg. 53131, Oct. 10 ,1997, effective Dec. 1, 1997; para. (d) revised, 63 Fed. Reg. 5734, Feb. 4, 1998, effective Feb. 4, 1998]

[2] The regulations at 37 C.F.R. §1.53(b) provide.

§1.53 Application number, filing date, and completion of application.

...

(b) Application filing requirements - Nonprovisional application. The filing date of an application for patent filed under this section, except for a provisional application under paragraph (c) of this section or a continued prosecution application under paragraph (d) of this section, is the date on which a specification as prescribed by 35 U.S.C. 112 containing a description pursuant to § 1.71 and at least one claim pursuant to § 1.75, and any drawing required by § 1.81(a) are filed in the Patent and Trademark Office. No new matter may be introduced into an application after its filing date. A continuing application, which may be a continuation, divisional, or continuation-in-part application, may be filed under the conditions specified in 35 U.S.C. 120, 121 or 365(c) and § 1.78(a).

(1) A continuation or divisional application that names as inventors the same or fewer than all of the inventors named in the prior application may be filed under this paragraph or paragraph (d) of this section.

(2) A continuation-in-part application (which may disclose and claim subject matter not disclosed in the prior application) or a continuation or divisional application naming an inventor not named in the prior application must be filed under this paragraph.

...

but implicit in the January 1999 petition is a request for Withdrawal of the Holding of Abandonment under 37 C.F.R. §1.181,[3] and vacation of the Notice of Abandonment mailed 4 December, 1998.

The petition is **GRANTED** in all regards.

There is no indication that petitioner herein was ever empowered to prosecute the instant application. If petitioner desires to receive future correspondence regarding this application, the appropriate power of attorney documentation must be submitted. A courtesy copy of this decision will be mailed to petitioner. However, all future correspondence will be directed to the address of record until such time as appropriate instructions are received to the contrary.

The instant record indicates that:

- the instant application was filed on 10 March, 1997;
- a "Notice of Missing Parts--Filing Date Granted" was mailed on 5 May, 1997, with a two- (2-) month response period and opportunity to obtain extension(s) of time;
- Petitioner filed as a response on 14 July, 1997, over a 9 July mail-date certificate, the petition to convert considered here with a check in the amount of $50.00, and included therewith a request and fee-authorization for an extension of time (one(1) month required), and such authority as necessary charge deficiencies to Deposit Account 13-2724;[4]
- the Notice of Abandonment was mailed on 4 December, 1998;
- Petitioner responded on 19 January, 1999, with a recounting of some of the facts set forth above and authorization for such fees as necessary to be charged to Deposit Account 50-0306.

The application is being forwarded to the Office of Initial Patent Examination:

- for further processing as a provisional application filed under 37 C.F.R.

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[3] The regulations at 37 C.F.R. §1.181 provide, in pertinent part:

§1.181 Petition to the Commissioner.

(a) Petition may be taken to the Commissioner: (1) From any action or requirement of any examiner in the *ex parte* prosecution of an application which is not subject to appeal to the Board of Patent Appeals and Interferences or to the court; (2) In cases in which a statute or the rules specify that the matter is to be determined directly by or reviewed by the Commissioner; and (3) To invoke the supervisory authority of the Commissioner in appropriate circumstances. * * *
(b) Any such petition must contain a statement of the facts involved and the point or points to be reviewed and the action requested. Brief or memoranda, if any, in support thereof should accompany or be embodied in the petition; and where facts are to be proven, the proof in the form of affidavits or declaration (and exhibits, if any) must accompany the petition.
(c) When a petition is taken from an action or requirement of an examiner in the *ex parte* prosecution of an application, it may be required that there have been a proper request for reconsideration (§1.111) and a repeated action by the examiner. The examiner may be directed by the Commissioner to furnish a written statement, within a specified time, setting forth the reasons for his decision upon the matters averred in the petition, supplying a copy thereof to the petitioner.
(d) Where a fee is required for a petition to the Commissioner the appropriate section of this part will so indicate. If any required fee does not accompany the petition, the petition will be dismissed. * * *
(f) Except as otherwise provided in these rules, any such petition not filed within 2 months from the action complained of may be dismissed as untimely. The mere filing of a petition will not stay the period for reply to an Examiner's action which may be running against an application, nor act as a stay of other proceedings.
* * *

[4] Although authorized, these charges never were made.

§1.53(c), not under 37 C.F.R. §1.53(b),[5] with a filing date of 10 March, 1997, using the application papers filed 10 March, 1997, and the authorization for fees filed on 14 July, 1997, and reaffirmed on 19 January, 1999, to pay fees as necessary; and

- for other processing as necessary consistent with this decision.

It is noted that charges have been made against Deposit Account 50-0306 as follows:

(a) for the full provisional filing fee, previously authorized but uncharged, in the amount of $150.00, with the deficient fee ($50.00) credited back to that account;

(b) the $110.00 fee for a one- (1-) month extension of time previously authorized but uncharged; and

(c) the $130.00 petition fee previously authorized but uncharged.

Telephone inquiries concerning this matter should be directed to Petitions Attorney John J. Gillon, Jr., at (703) 305-9199.

Brian Hearn
Petitions Examiner
Office of Petitions
Office of the Assistant Commissioner
for Patent Examination Policy

cc:
SARAH B. ADRIANO
MANDEL & ADRIANO
35 NORTH ARROYO PARKWAY/STE. 60
PASADENA, CA 91103

[5] The regulations at 37 C.F.R. §1.53(b) provide:

§1.53 Application number, filing date, and completion of application.

...

(b) Application filing requirements - Nonprovisional application. The filing date of an application for patent filed under this section, except for a provisional application under paragraph (c) of this section or a continued prosecution application under paragraph (d) of this section, is the date on which a specification as prescribed by 35 U.S.C. 112 containing a description pursuant to § 1.71 and at least one claim pursuant to § 1.75, and any drawing required by § 1.81(a) are filed in the Patent and Trademark Office. No new matter may be introduced into an application after its filing date. A continuing application, which may be a continuation, divisional, or continuation-in-part application, may be filed under the conditions specified in 35 U.S.C. 120, 121 or 365(c) and § 1.78(a).

(1) A continuation or divisional application that names as inventors the same or fewer than all of the inventors named in the prior application may be filed under this paragraph or paragraph (d) of this section.

(2) A continuation-in-part application (which may disclose and claim subject matter not disclosed in the prior application) or a continuation or divisional application naming an inventor not named in the prior application must be filed under this paragraph.

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